10/687,578

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(FILE 'HOME' ENTERED AT 18:54:20 ON 12 JAN 2006)

FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH, LIFESCI' ENTERED AT 18:55:07 ON 12 JAN 2006

L1 296 S RECEPTOR(3A)ADVANCED(W)GLYCATION(W)ENDPRODUCT
L2 251450 S RESTENOSIS OR STENOSIS
L3 3 S L1(S)L2
L4 9 S L1 AND L2
L5 3 DUP REM L3 (0 DUPLICATES REMOVED)
L6 5 DUP REM L4 (4 DUPLICATES REMOVED)

=> d bib ab 1-5 l6

L6 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2003:804087 CAPLUS
DN 140:121912
TI Blockade of **receptor** for **advanced glycation endproducts**: a new target for therapeutic intervention in diabetic complications and inflammatory disorders
AU Hudson, Barry I.; Bucciarelli, Loredana G.; Wendt, Thoralf; Sakaguchi, Taichi; Lalla, Evanthia; Qu, Wu; Lu, Yan; Lee, Larisse; Stern, David M.; Naka, Yoshifumi; Ramasamy, Ravichandran; Yan, Shi Du; Yan, Shi Fang; D'Agati, Vivette; Schmidt, Ann Marie
CS College of Physicians and Surgeons, Departments of Surgery, Pathology and Medicine, Columbia University, New York, NY, 10032, USA
SO Archives of Biochemistry and Biophysics (2003), 419(1), 80-88
CODEN: ABBIA4; ISSN: 0003-9861
PB Elsevier Science
DT Journal; General Review
LA English
AB A review. The glycation and oxidation of proteins/lipids leads to the generation of a new class of biol. active moieties, the advanced glycation endproducts (AGEs). Recent studies have elucidated that carboxymethyllysine (CML) adducts of proteins/lipids are a highly prevalent AGE in vivo. CML-modified adducts are signal transduction ligands of the receptor for AGE (RAGE), a member of the Ig superfamily. Importantly, CML-modified adducts accumulate in diverse settings. In addition to enhanced formation in settings of high glucose, these adducts form in inflammatory milieu. Studies performed both in vitro and in vivo have suggested that the proinflammatory/tissue destructive consequences of RAGE activation in the diabetic/inflamed environment may be markedly attenuated by blockade of the ligand-RAGE axis. Here, we will summarize the known consequences of RAGE activation in the tissues and highlight novel areas for therapeutic intervention in these disease states.
RE.CNT 72 THERE ARE 72 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2006 ACS on STN
AN 2002:293614 CAPLUS
DN 136:304083
TI A method for inhibiting new tissue growth in blood vessels in a patient subjected to blood vessel injury
IN Stern, David M.; Schmidt, Ann-Marie; Marso, Steven; Topol, Eric; Lincoff, A. Michael
PA The Trustees of Columbia University In the City of New York, USA
SO PCT Int. Appl., 43 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1
PATENT NO. KIND DATE APPLICATION NO. DATE

PI	WO 2002030889	A2	20020418	WO 2001-US32036	20011012
	WO 2002030889	A3	20020711		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2002013192	A5	20020422	AU 2002-13192	20011012
PRAI	US 2000-687528	A	20001013		
	WO 2001-US32036	W	20011012		

AB This invention provides for a method for inhibiting new tissue growth in blood vessels in a subject, wherein the subject experienced blood vessels injury, which comprises administering to the subject a pharmaceutically effective amount of an inhibitor of **receptor** for **advanced glycation endproduct** (RAGE) so as to inhibit new tissue growth in the subject's blood vessels. The invention also provides for method for inhibiting neointimal formation in blood vessels in a subject, wherein the subject experienced blood vessel injury, which comprises administering to the subject a pharmaceutically effective amount of an inhibitor of **receptor** for **advanced glycation endproduct** (RAGE) so as to inhibit neointimal formation in the subject's blood vessels. The invention also provides a method for preventing exaggerated **restenosis** in a diabetic subject which comprises administering to the subject a pharmaceutically effective amount of an inhibitor of **receptor** for **advanced glycation endproduct** (RAGE) so as to prevent exaggerated **restenosis** in the subject. In the example provided, a significant reduction in neointimal area was observed in fatty Zucker rats treated with soluble **receptor** for **advanced glycation endproduct** following carotid artery injury.

L6 ANSWER 3 OF 5 MEDLINE on STN
AN 2002199309 MEDLINE
DN PubMed ID: 11931721
TI **Receptor** for **advanced glycation endproducts** (RAGE) and vascular inflammation: insights into the pathogenesis of macrovascular complications in diabetes.
AU Wendt Thoralf; Bucciarelli Loredana; Qu Wu; Lu Yan; Yan Shi Fang; Stern David M; Schmidt Ann Marie
CS Division of Surgical Science, Department of Surgery, Columbia University College of Physicians & Surgeons, 630 West 168th Street, P&S 17-401, New York, NY 10032, USA.
SO Current atherosclerosis reports, (2002 May) 4 (3) 228-37. Ref: 52
Journal code: 100897685. ISSN: 1523-3804.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200306
ED Entered STN: 20020405
Last Updated on STN: 20021211
Entered Medline: 20030610
AB The incidence and severity of atherosclerosis is increased in patients with diabetes. Indeed, accelerated macrovascular disease in diabetic patients has emerged as a leading cause of morbidity and mortality in the

United States and worldwide. Multiple investigations have suggested that there are numerous potential contributory factors that underlie these observations. Our laboratory has focused on the contribution of **receptor** for **advanced glycation endproducts** (RAGE) and its proinflammatory ligands, advanced glycation endproducts (AGEs) and S100/calgranulins in vascular perturbation, manifested as enhanced atherogenesis or accelerated **restenosis** after angioplasty. In rodent models of diabetic complications, blockade of RAGE suppressed vascular hyperpermeability, accelerated atherosclerotic lesion area and complexity in diabetic apolipoprotein E-deficient mice, and prevented exaggerated neointimal formation in hyperglycemic fatty Zucker rats subjected to injury of the carotid artery. In this review, we summarize these findings and provide an overview of distinct mechanisms that contribute to the development of accelerated diabetic macrovascular disease. Insights into therapeutic strategies to prevent or interrupt these processes are presented.

L6 ANSWER 4 OF 5 MEDLINE on STN DUPLICATE 1
AN 2001227980 MEDLINE
DN PubMed ID: 11257120
TI The high mobility group (HMG) boxes of the nuclear protein HMG1 induce chemotaxis and cytoskeleton reorganization in rat smooth muscle cells.
AU Degryse B; Bonaldi T; Scaffidi P; Muller S; Resnati M; Sanvito F; Arrigoni G; Bianchi M E
CS Department of Genetics and Microbiology, University of Milan, 20133 Milan, Italy.. degryse@scripps.edu
SO Journal of cell biology, (2001 Mar 19) 152 (6) 1197-206.
Journal code: 0375356. ISSN: 0021-9525.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200104
ED Entered STN: 20010502
Last Updated on STN: 20021218
Entered Medline: 20010426
AB HMG1 (high mobility group 1) is a ubiquitous and abundant chromatin component. However, HMG1 can be secreted by activated macrophages and monocytes, and can act as a mediator of inflammation and endotoxic lethality. Here we document a role of extracellular HMG1 in cell migration. HMG1 (and its individual DNA-binding domains) stimulated migration of rat smooth muscle cells in chemotaxis, chemokinesis, and wound healing assays. HMG1 induced rapid and transient changes of cell shape, and actin cytoskeleton reorganization leading to an elongated polarized morphology typical of motile cells. These effects were inhibited by antibodies directed against the **receptor** of **advanced glycation endproducts**, indicating that the **receptor** of **advanced glycation endproducts** is the receptor mediating the HMG1-dependent migratory responses. Pertussis toxin and the mitogen-activated protein kinase kinase inhibitor PD98059 also blocked HMG1-induced rat smooth muscle cell migration, suggesting that a G(i/o) protein and mitogen-activated protein kinases are required for the HMG1 signaling pathway. We also show that HMG1 can be released by damage or necrosis of a variety of cell types, including endothelial cells. Thus, HMG1 has all the hallmarks of a molecule that can promote atherosclerosis and **restenosis** after vascular damage.

L6 ANSWER 5 OF 5 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
AN 2002:264313 BIOSIS
DN PREV200200264313
TI **Restenosis**: Central role of RAGE-dependent neointimal expansion.
AU Sakaguchi, Taichi [Reprint author]; Sousa, Monica [Reprint author]; Yan,

Shi Du [Reprint author]; Yan, Shi-Fang [Reprint author]; Duda, Stephan; Arnold, Bernd; Nawroth, Peter P.; Schmidt, Ann Marie; Stern, David M.; Naka, Yoshifumi

CS Columbia Univ, New York, NY, USA

SO Circulation, (October 23, 2001) Vol. 104, No. 17 Supplement, pp. II.522-II.523. print.
Meeting Info.: Scientific Sessions 2001 of the American Heart Association. Anaheim, California, USA. November 11-14, 2001. American Heart Association.
CODEN: CIRCAZ. ISSN: 0009-7322.

DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 1 May 2002
Last Updated on STN: 1 May 2002

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